Remarks
ON
EARLY SIGNS OF CARDIAC FAILURE OF THE CONGESTIVE TYPE.*

BY
SIR THOMAS LEWIS, M.D., F.R.C.P., F.R.S.,
PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

To the student of cardio-vascular disease the ability to form a broad estimate of the way in which the general circulation is carried on under various circumstances is of paramount importance. When a patient seeks advice, and disease of the heart is suspected or is present, one question should stand prominently in the foreground, namely, What is the capacity of the heart for work? The answer to this question dominates both prognosis and treatment to an extent that forces other considerations well into the background in most cases of chronic heart disease. Innumerable tests have been, and continue to be, devised to answer this question; they are based mainly upon pulse and blood pressure readings taken in various circumstances of posture, exercise, etc. One danger of these tests is that, as they are frequently presented by means of formulae, they are apt to create an impression of scientific accuracy that they are far from possessing. Experience of such tests has convinced me that they have little practical value, and, tending as they often do to becomes rules of thumb, they likewise become most undesirable. Recently, numerous attempts have been made to estimate cardiac output in terms of fluid volume; while these methods have a significance in physiology, they have as yet found no application in clinical work, and are unlikely to find such application for many years to come.

Cardiac capacity in patients is to be gauged rather by a close study of the symptoms arising out of exercise and by the early signs of congestion; by simple methods that can be grasped and applied by practitioners; methods which for these reasons, as well as for their proved worth, stand unrivalled in practical everyday work.

If, in speaking of heart failure, we are to include not only the terminal phenomena but also all the earlier manifestations that may be linked transitionally with them, then we shall review, if only briefly, the main results of cardiac muscle weakness in its different grades. It has been held, and not without justification, that cardiac failure can be of two distinct forms, namely, that manifesting itself chiefly by the well-known symptoms belonging to the anginal group, and that manifesting itself first in breathlessness, and continuing in its natural development to profound engorgement of the venous system. It is not proposed to cover all this ground, but to deal only with failure of the second type, a type that ends in what is often spoken of as "loss of compensation" or "decompensation," two terms which, in my view, should be abandoned, since, in so far as they retain their original meaning, they infer (and infer unnecessarily) far more than can be proved, and since they conceal the essence of the situation; I prefer and advocate the term "congestive failure."

When congestive failure develops gradually, the symptoms and signs of this condition come insidiously, and the order of their appearance is definite. The main symptom is breathlessness. All men and women, however athletic, become breathless on exercise; the first indication of cardiac failure of this type manifests itself in a limitation of the field of response; an active subject notices breathlessness or unusual breathlessness on undertaking some customary act of vigorous work. As this failure progresses, the same breathlessness continues to be experienced, but it is experienced in response to an amount of work that decreases as time passes. This progression may be more or less continuous, spread over months or years, or it may occur in definite steps; this latter is by far the commoner. In grading cardiac breathlessness I do so in the first instance, not by the degree of distress it occasions, but by the amount of physical effort that brings obvious breathlessness into being. Breathlessness occurs on walking quickly or uphill, on walking on the flat, on walking slowly short distances; later it begins to be present when at rest; these are the grades. *It is in the last two stages that the early signs of actual venous congestion appear, and as congestion increases, breathlessness at rest becomes more obvious and more distressing.* Thus the complete course from health to gross heart failure is displayed by the cardinal symptom, breathlessness, graded in its degree from normal breathlessness on active exertion to constant distress when the body rests completely. These grades of breathlessness are ascribed to various deficiencies in the flow of aerated blood through the brain; at first the deficiency is confined to those exercises in which normally the cardiac output is much above resting value; at last there is a deficiency in the physiological quantity of blood expelled by the heart while the body is at rest. It is then that blood begins to collect on the venous side and the patient begins to manifest signs of congested veins, and, associated with these, such signs as enlargement of the liver, cyanosis, a high-coloured scanty urine, ascites, dropsy of the lower members, and the signs of congested and oedematous lungs. Of paramount importance, however, are the signs in the veins; that is so because they are usually amongst the earliest that can be detected; for that reason every medical student should make a full study of them.

Direct measurement of venous pressure has been much used in my wards in past years, and gives in my experience a good idea, within its limits, of the patient's state; it presents, in repeated readings, clear indications of the patient's course. But it is obvious that this method cannot come into wide use, so for many years I have striven to displace it by simple bedside tests that may be applied by any thoughtful and good observer. In arriving at the clinical methods now used in my department, actual manometric determinations of venous pressure have formed important and frequent guides. After describing quite briefly the manometric method, and having illustrated the simple hydrostatic principles involved, the bedside signs that have been found of value will be discussed.

Measures of Venous Pressure.

To measure pressure in the veins more than one device has been employed. A simple piece of apparatus (after the original plan of Moritz) consists of a manometer connected to a wide needle by rubber tubing, the whole filled with a solution of sodium citrate to prevent clotting. The needle is driven into a vein, and the height at which the fluid comes to rest in the manometer measures the venous pressure in centimetres of citrate solution. This, broadly described, is a method of accuracy. A vertical measure, taken from the meniscus to the point at which the vein is punctured, tells the pressure *in the vein at that point.* The measure serves also as a gauge of pressure in the right auricle, provided that no local impediment to the stream exists in the venous stem between the point chosen and the right auricle, the veins being widely patent, and provided that the level of the venous point chosen relative to the heart is known. For if the vein chosen is in the arm, and the arm is raised, the pressure in that vein will fall, or if the arm is lowered it will rise, correspondingly. Since the position of the punctured point, relative to the general system of veins, will vary in different circumstances, it is customary to choose some standard point of reference, and the point we have chosen in patients lying supine is the lower border of the manubrium sterni. If the needle is passed into the median basilic vein of a normal subject so postured, the fluid column in the manometer will usually come to rest level with the sternum, or a few centimetres below, or, less commonly, a centimetre or two above it. Thus, if the punctured vein lies level with the sternum, the meniscus will come to rest at or about the level of the puncture, and, as the arm is depressed, it will not move appreciably relative to the sternum, but the column will

* The gist of a lecture delivered before the Portsmouth Division of the British Medical Association on December 13th, 1928.

reach above the vein by the amount the arm is lowered (Fig. 1). The venous reservoir thus behaves much like a simple reservoir. In this the surface of fluid lies at a given level, and there the pressure is atmospheric or zero. In the depths of the reservoir pressure is greater, and it is greater by the amount of the vertical column of fluid that extends from any given point to the surface of the fluid.

We may speak of the zero (or atmospheric) pressure level of the fluid in the venous system in much the same sense, and that level is near the lower border of the manubrium sterni in normal subjects. This point of reference is chosen because it represents approximately the level of zero pressure whether the body is horizontal or vertical, or in any intermediate position. It is likewise true of the flaccid body that the pressure in veins lying at different levels below the sternum is greater, and at levels above the sternum is less, than atmospheric pressure. Thus, normally, all veins which lie higher than the manubrial point are collapsed, all which lie below it are distended. If, therefore, we can gauge the precise level at which the veins collapse we have a gauge of the filling of the venous reservoir and of general, or to be more exact of right auricular venous, pressure. A simple and old test of venous zero level is to watch the veins on the back of the hand or at the elbow. These, when the arm hangs limply by the side, are swollen; let the arm be lifted in its flaccid state, while the veins are watched, and, in normal resting people, these will flatten as the hand comes to the height of the manubrium. The arm must be lifted passively; it must not lift itself, for active movement brings the muscles of the limb into play, and these press upon the veins and disturb the test. Another and better gauge is to place the subject upon his back, the head resting upon pillows (Fig. 2). The external jugular veins are then usually to be seen as swollen vessels in the neck, but, as they are traced upwards, the swelling ends; it ends at a point that represents atmospheric pressure in the veins, and in normal people this is at a point of the neck that is level with the sternum, or a little higher or a little lower.

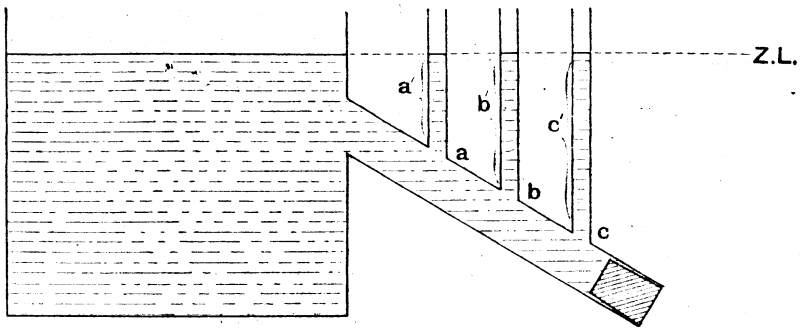

FIG. 1.—A reservoir of water has a wide pipe joined to it. If manometers are attached at *a*, *b*, *c*, the measured pressure above these points is represented by the increasing size of the columns *a'*, *b'*, *c'*, but all these columns rise to the same zero level (Z.L.). All indicate equally well the height of the water in the reservoir.

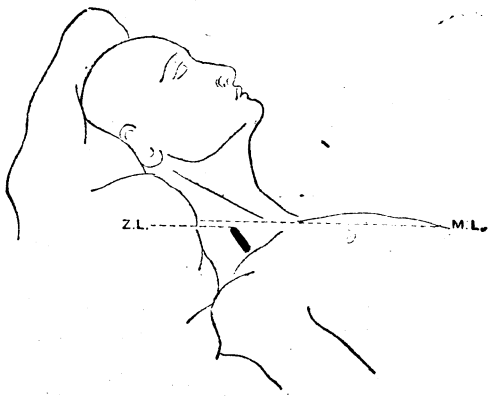

FIG. 2.—Normal subject supine with head on pillows. The zero level (Z.L.), indicated by the top point of swelling of the external jugular vein, lies a little below the manubrial line (M.L.).

It is necessary to be sure that the swelling ends where it seems to in the external jugular, and that the vein is not merely running deeper in the higher part of its course. To find out, press a finger lightly on the vein below and it will at once fill in its length and show its whole superficial course. The natural swelling of the neck veins will extend perhaps a third of the way to the jaw, more or less, according to the inclination of the neck to the body. Lift the foot of the couch and in the neck the blood tide advances; lift the head of the couch and it recedes. In these acts think of the surface of the venous pool as you would think of the level of fluid in a long pan of water, lifted at one end or at the other; watch the rise and fall so that you may recognize the surface and know the general level (Fig. 3).

The venous zero level rises to various heights in congestion. Where venous pressure is raised a little the column is seen to stand in the neck of the supine subject as high as the centre of the sterno-mastoid muscle or beyond this; it comes unmistakably above the sternal level on both sides of the neck. Where pressure is greater, the veins are swollen to the jaw. Note especially that it is not the anatomical point reached in the neck that matters, but the vertical distance above the sternum. The former is affected by the inclination of the neck to the body, etc.

Where pressure is higher still, the veins remain swollen throughout the neck when the head and shoulders are lifted off the bed. The gauge remains unaltered; it is the vertical height to which the swollen veins extend, or may be raised above the sternum without collapsing. The veins are never full in the neck in a normal unclothed person standing easily in ordinary circumstances in the erect posture; in conspicuous congestion they run the length of the neck like cords.

Thorough familiarity with these phenomena and with the very simple hydrostatic principle involved is an essential matter. It is to be observed that the venous system with its many tributaries is treated as a system of simple and open tubes. The tests should be carried out deliberately, with due precautions; more especially is it to be ensured that no article of clothing or tense muscle can press upon the veins between the point of observation and the heart. The subject must be stripped, and the parts concerned must be rendered flaccid, and must be so placed that their position is favourable to venous emptying. Sometimes the veins of the neck are at first tense, presumably because their passage through the deep fasciae of the neck lacks freedom; a little passive movement of the head will then open up the way and truer estimates will be obtained. Veins swollen on one side only point to local obstruction of their immediate outlet and have no value from our present standpoint; often such swelling can be released by a little rotation of the neck; the vein that has the lowest pressure is alone to be used as an index of general venous pressure.

A rise of venous zero level in the neck so gauged may be confirmed by gentle palpation of the visible veins, to estimate the tension within them.

The gauges of venous zero level, somewhat less accurate perhaps than manometric readings, are quite exact enough for clinical work, and should be used in part or in whole as a routine in cases of real or suspected disease of the cardio-vascular system. The tests in the neck are preferred to those in the hand or arm, for several reasons. Thus there is less likelihood both of obstruction to, and of muscular emptying of, a cervical vein than there is of a limb vein, since the course is shorter and muscular flaccidity is more readily ensured along this course. Another and important reason is that sclerosis of the veins of the arm is not infrequent; and sclerosis must not be overlooked, as it interferes seriously with collapse; it is readily identified by feeling the emptied vessel. In the legs, the veins, by middle life, and especially the more visible ones over the ankles, are usually sclerosed. In most cases venous pressure in the leg is most difficult to ascertain clinically. In general it may be said that the tests are the more reliable the nearer the vein is to the heart.

I have dealt so far with estimates of the venous zero level by the method of visible collapse. This is the usual method, but there are instances in which no suitable vein in the neck is visible, and here another method is adopted.

The Venous Pulse as an Index.

Not infrequently the position of the venous pulse forms the most reliable sign of normal or raised pressure. A venous pulse at the root of the neck is a normal phenomenon in the supine subject; it is not pathological even if free. In young people this venous pulse commonly shows a great excursion, and is often, though erroneously, regarded as abnormal or is confused with arterial pulsation. To be able to distinguish between the two with certainty is important; it is not difficult, yet there are few who have learnt the ways. In many subjects venous pulsation is obvious at a glance, because it involves superficial veins such as the external jugular, and is evidently within them; in others, and especially those in whom no superficial veins are visible, discrimination is required. Venous pulsation, in the supine and where there is no congestion, involves usually the lower part of the neck along the line of the carotid sheath and subclavian vessels; it is occurring in the internal jugular and subclavian veins, and therefore has a main distribution not dissimilar to that of the chief arteries, but, owing to large tributaries in the subclavian triangle, it is here more diffuse and superficial than is pulsation of the arteries.

The commonest large pulsation in the neck comes from the internal jugular. It frequently extends to and moves the lobe of the ear, and is then usually mistaken for carotid pulsation, a serious mistake to make. Venous pulsation is rarely or barely palpable, while a visible arterial pulse is always felt; the testing finger should be laid as lightly as possible on the pulsating skin, lest by deep pressure the beating of an underlying artery is made perceptible. Venous pulsation is a complex and fluctuating, or a slow and welling movement; it does not jerk sharply to a sustained thrust as does an artery. Such in general are the signs of venous pulsation in the uncongested. In the congested they are modified; more especially the field of venous pulsation is displaced. That is so because *the maximal venous pulsation occurs in veins on the point of collapse*; it is imperceptible in collapsed veins or in veins tightly distended. Thus, in the normal supine subject, the venous pulse occurs in the lowest veins of the neck, for these are just full; it is unseen or lacks prominence in the higher veins of the neck, and that is true of all the cervical veins when the subject is erect. The same change of posture renders carotid pulsation more distinct and brings it particularly into prominence in the carotid triangle, where the artery is most superficial. When much congestion is present and the subject is supine, the veins of the neck are often too tense to pulsate; tip the patient into a more erect, or bring him to a completely erect, posture, and the level of zero venous pressure falls in the neck veins, the tide recedes, and near its margin there appears abundant pulsation. In these patients venous pulsation is often extensive, spreading to the veins of the upper arm and even forearm, and in these parts the association between pulsation and a condition of filling bordering on collapse is often displayed very clearly. Here sometimes, but more frequently in the neck, the level of pulsation is seen to alter with respiration, usually changing to a point nearer the heart in inspiration, because then the venous pressure falls. If this respiratory movement interferes with estimates it can be suspended.

It will be seen from what has been said that the level of maximal venous pulsation approaches closely to the level of zero venous pressure; thus it can be used to confirm the estimate of the point of collapse; it forms the sole gauge when large superficial veins are absent or invisible; in fact, this is the chief bedside value of visible venous pulsation. When veins pulsate freely above the level of the manubrial line they are overfull. Again from this standpoint a thoughtful examination is desirable, for the cervical veins may be overfull, not only because the venous system as a whole is replete with blood, but temporarily because pressure in a part of it is raised by muscular action. Thus, in many normal subjects, if they lean forward as they sit, prominent pulsation appears in the jugular veins; that is so because pressure is exerted in this posture on the abdominal reservoir and blood is displaced to the neck. It can be so displaced artificially in subjects in the supine posture by pressing with the hand on the abdomen; the act tends to flood the neck and to increase pulsation in it. The degree of flooding so occurring is sometimes helpful in estimating the amount of blood held in the abdomen, and in helping to determine the sizes and positions of the cervical veins. Lastly, it is to be said that in congestion the venous pulse tends to lose its fluctuating character, becomes more sustained, and sometimes becomes plainly palpable. In such patients, where the distinction between arterial and venous pulses is sometimes less easy, and depends mainly upon the distribution of the pulse and upon actual visibility or otherwise in superficial veins, the remaining signs of a congested venous system are always manifest.

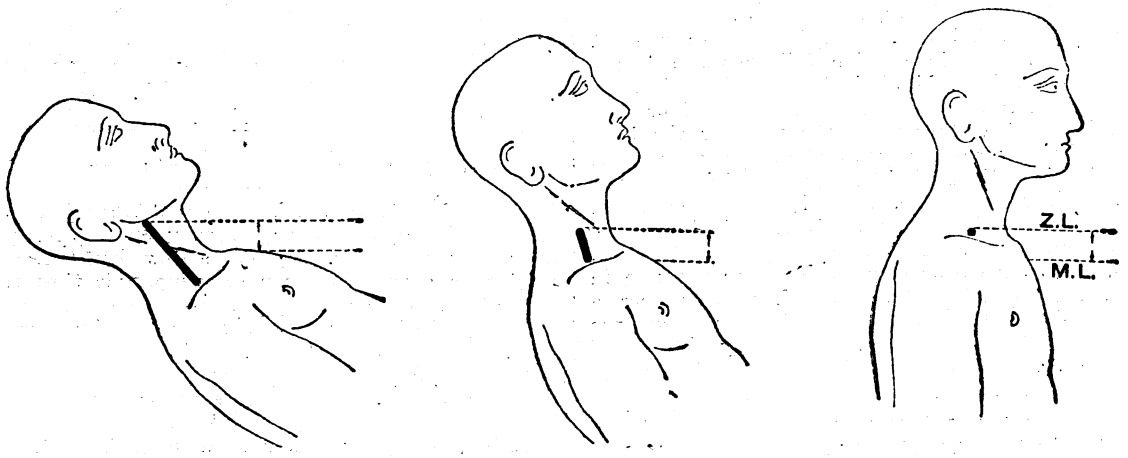

FIG. 3.—A diagram illustrating excessive venous pressure equivalent to about 8 cm. of water. In the upright position the zero level may be just a little above the clavicle; as the subject is inclined the zero level comes to occupy a higher and higher position in the neck until it reaches to the angle of the jaw. Its vertical distance above the manubrium does not change much.

Other Signs of Congestion.

Of the remaining signs associated with venous congestion I propose to say nothing except of those found in the liver, for here also important early signs are found. The firm smooth margin of a congested liver is generally palpable; it is felt at varying levels between the rib margin and the navel, and eventually may lie even lower than the last. Failure to palpate the edge of a congested liver is usually the result of beginning to feel the abdomen at too high a level; the whole hand should first be placed, flat and transversely, across the abdomen, well below the navel with gentle yet firm pressure, and gradually worked

upwards until resistance is first felt. If the patient then breathes in and out freely the moving edge of the organ is rarely missed. Percussion is also most helpful; I prefer to use it in the mid-line and of moderate intensity; normally, in these circumstances, the abdomen is resonant to the midriff, for the thin margin of the liver, where it crosses the intercostal angle, fails materially to modify the note, unless the liver edge lies low. Using these methods, early congestion of the liver is generally detectable without difficulty, unless the abdominal wall is unduly thick or tense.

The signs in the veins and in the liver I have emphasized for a chief reason—namely, because they are displayed in early stages of congestion. When the full and classical signs of failure are present, it requires neither close observation nor much discrimination to know it. The diagnosis of early congestion is more difficult, and in the long run it is much more important. It can be accomplished by careful study of the veins and of the liver, and by these means only. Experience teaches that to rely on a single sign is precarious. Compare this sign and that, and confident recognition of the patient's state grows as these signs fall together to form a harmonious picture. Thus, on finding unquestionable evidence of engorged veins in neck, or neck and arms, we look at once at the liver; if we find equally unquestionable evidence that this organ is normal, we know that we are dealing with a local obstruction of the veins. Incidentally, in this connexion observe that veins so obstructed cannot be induced to pulsate. Suppose, however, that in cardiac failure the evidence derived from the veins is indistinct, that there is some doubt whether their pressure exceeds the normal or does not—and this doubt will come to the most or the least experienced observer according to the type of patient under examination—then distinct though slight enlargement of the liver becomes a most important evidence. Generally speaking, enlargement of the liver in its several degrees goes hand-in-hand with engorgement of the veins. In general, the liver does not enlarge before the rise in pressure in the veins can be detected, neither does a distinct venous engorgement manifest itself without the liver edge descending appreciably. There may be cases of difficulty where, owing to fascial obstruction of the upper veins, or enlargement of the liver from causes other than congestion, or failure of the liver to enlarge in congestion owing to cirrhosis, a discord appears. There is also the case in which engorgement of the liver has been present for a very long time; in such, even if the signs of increased pressure in the veins greatly decline, the size of the liver may not decrease much or at least proportionately. But in most instances harmony is found and brings an element of certainty in estimating the degree of failure, and this is so even in regard to early diagnosis. To correlate, and to see accord, in estimating signs is of the utmost consequence, and in this connexion I would emphasize another and most important correlation. *Patients who suffer from general congestion of the venous system are without exception breathless, either at complete rest or upon very slight exertion.* If, therefore, a subject is thought on examination to manifest the early signs of venous stasis, and yet it is clear that breathlessness is not experienced even with quiet or moderate exercise, the opinion first formed must be revised; for it is incorrect. Moreover, there is a perfectly clear relation, constant within narrow limits, between the degree of breathlessness and the pressure in the veins; the relation is so clear that *if it is established that a patient has no congestion of the venous system and is yet breathless in bed, then that breathlessness is not primarily cardiac in origin.* The proper use of this knowledge will save a very large number of serious diagnostic blunders; all that is required is that the point of venous collapse should be proved to occur in any cervical vein at or below the level of the manubrium.

In conclusion, I would repeat that an understanding of cardiac failure of the congestive type is obtainable only by those who truly appreciate the manner in which the symptoms on the one hand and the signs on the other become linked together; the subjective and the objective manifestations are part and parcel of one process, and are dependent on the same fundamental causes, namely, loss of cardiac reserve.

Those who desire efficiently to manage chronic cardiac cases should observe and consider very carefully how little is really gained, and how seldom there is any gain, by repeated auscultation of the heart sounds. Signs so obtained rarely change, they are not signs that indicate improvement or deterioration; but the symptoms and signs upon which emphasis has here been laid do change frequently in one or two directions. They form the chief indications of the course the patient is pursuing and will pursue, for they tell how the blood is circulating in the body generally under various conditions and speak of the capacity of the heart to do work. Those in charge of cardiac patients will do well to acquaint themselves fully with the very definite and sensitive symptoms and the clear signs of early failure here discussed, and to familiarize themselves with the venous manifestations as these occur both in disease and in health.

To understand the phenomena one by one as they are written down and explained is not difficult; to think that such understanding can be brought at once into full play in practical work is to underestimate the situation and to fail in the application; a full grasp and working knowledge can be attained only by diligent observation and thought in which the abnormal is accurately weighed against and considered with the normal.

MYELOMATOSIS, OR BENCE-JONES PROTEINURIA.

BY

A. MACBETH ELLIOT, M.D.

IN November, 1845, Dr. Watson sent a specimen of urine to Bence-Jones, with the question "What is it?" written on a tag of paper. Within a few hours Bence-Jones received a second specimen of urine, this time from Dr. MacIntyre. Both specimens were from the same patient, a man who had been under Watson's care since the previous May. MacIntyre had been called in as consultant.

The man, a well-to-do grocer aged 47, had been ailing for over a year. When first seen by MacIntyre he was thin, emaciated, sallow, pale; his expression was that of suffering, though when not in pain he was cheerful. Pressure along the spine produced little discomfort, but if the pelvis was pressed he complained of severe pain. The arms and legs could be moved freely without any discomfort. Bence-Jones saw the man alive once only, but he received many samples of his urine. The man died on January 2nd, 1846.

In 1847 Bence-Jones read a paper before the Royal Society. After repeated tests and much deep thought, he had arrived at the following conclusions concerning the peculiar deposit in the patient's urine: (1) that the precipitate was an oxide of albumin, and he was satisfied that it was the hydrated deutoxide; (2) that the peculiar characteristic of this hydrated deutoxide was its solubility in boiling water, the precipitate thrown down with nitric acid disappearing on boiling to reappear on cooling. In 1850 MacIntyre published his clinical notes on the case. In the *Dublin Journal* of 1846 Dalrymple had given a full pathological report of the case under the title of "mollities ossium."

I have been unable to trace any further report of a similar nature till the year 1889, when Kahler pointed out the difference between this condition and osteomalacia. Bradshaw, in 1897, described a case, and reported further on it in 1906. Moffat wrote fully on this disease in 1905, and since then there have been various cases reported.

Martin Solon, in his *Traitement d'albuminurie* (Paris, 1838), notes that in one of his cases he came across the peculiar fact that when the patient's urine was treated by nitric acid or heat there was a precipitate which disappeared on boiling; he should therefore be credited with having been the first to draw attention to this peculiar urinary deposit, since apparently it was the same as that found by Watson and MacIntyre.

In October of 1925 a woman was brought up from the country to see me; I was told that it was a case of acute